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DATE: Tuesday, January 10, 2006

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 NEWS 18  DEC 21 IPC search and display fields enhanced in CA/CAplus with the
                 IPC reform
 NEWS 19  DEC 23 New IPC8 SEARCH, DISPLAY, and SELECT fields in USPATFULL/USPAT2

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               AND CURRENT DISCOVER FILE IS DATED 19 DECEMBER 2005.
               V8.0 USERS CAN OBTAIN THE UPGRADE TO V8.01 AT
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FILE 'HOME' ENTERED AT 09:19:28 ON 10 JAN 2006

=> file medline
COST IN U.S. DOLLARS SINCE FILE ENTRY TOTAL SESSION
FULL ESTIMATED COST 0.63 0.63

FILE 'MEDLINE' ENTERED AT 09:21:17 ON 10 JAN 2006

FILE LAST UPDATED: 7 JAN 2006 (20060107/UP). FILE COVERS 1950 TO DATE.

On December 11, 2005, the 2006 MeSH terms were loaded.

The MEDLINE reload for 2006 will soon be available. For details on the 2005 reload, enter HELP RLOAD at an arrow promt (=>). See also:

http://www.nlm.nih.gov/mesh/
http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html
http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_med_data_changes.html
http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_2006_MeSH.html

OLDMEDLINE is covered back to 1950.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2006 vocabulary.

This file contains CAS Registry Numbers for easy and accurate

=> s tu GnRH or gonadotropin(w)releasing(w)hormone
1145754 TU
204 TUS
1145918 TU
(TU OR TUS)
11745 GNRH
107 GNRHS
11747 GNRH
(GNRH OR GNRHS)
0 TU GNRH
(TU(W)GNRH)
41701 GONADOTROPIN
22719 GONADOTROPINS
55507 GONADOTROPIN
(GONADOTROPIN OR GONADOTROPINS)
57465 RELEASING
268964 HORMONE
177879 HORMONES
389158 HORMONE
(HORMONE OR HORMONES)
7655 GONADOTROPIN(W)RELEASING(W)HORMONE
L1 7655 TU GNRH OR GONADOTROPIN(W)RELEASING(W)HORMONE

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=> s l1 and Kaposi(w)sarcoma
         10558 KAPOSI
            40 KAPOSIS
         10568 KAPOSI
                 (KAPOSI OR KAPOSIS)
         70545 SARCOMA
         11081 SARCOMAS
           119 SARCOMATA
         73808 SARCOMA
                 (SARCOMA OR SARCOMAS OR SARCOMATA)
          1041 KAPOSI(W)SARCOMA
L2           0 L1 AND KAPOSI(W)SARCOMA

=> s l1 and glioblastoma
         11148 GLIOBLASTOMA
          2325 GLIOBLASTOMAS
         12012 GLIOBLASTOMA
                 (GLIOBLASTOMA OR GLIOBLASTOMAS)
L3           0 L1 AND GLIOBLASTOMA

=> s l1 and medulloblastoma
          4566 MEDULLOBLASTOMA
          1170 MEDULLOBLASTOMAS
          4907 MEDULLOBLASTOMA
                 (MEDULLOBLASTOMA OR MEDULLOBLASTOMAS)
L4           6 L1 AND MEDULLOBLASTOMA

=> dup rem
ENTER L# LIST OR (END):l4
PROCESSING COMPLETED FOR L4
L5            6 DUP REM L4 (0 DUPLICATES REMOVED)

=> dis ibib abs l5 1-6

L5   ANSWER 1 OF 6     MEDLINE on STN
ACCESSION NUMBER:    2005069367     MEDLINE
DOCUMENT NUMBER:     PubMed ID: 15562029
TITLE:               Differential role of progesterone receptor isoforms in the
                     transcriptional regulation of human gonadotropin-
                     releasing hormone I (GnRH I) receptor,
                     GnRH I, and GnRH II.
AUTHOR:              An Beum-Soo; Choi Jung-Hye; Choi Kyung-Chul; Leung Peter C
                     K
CORPORATE SOURCE:    Department of Obstetrics and Gynecology, University of
                     British Columbia, Vancouver, British Columbia, Canada V6H
                     3V5.
SOURCE:              Journal of clinical endocrinology and metabolism, (2005
                     Feb) 90 (2) 1106-13.  Electronic Publication: 2004-11-23.
                     Journal code: 0375362. ISSN: 0021-972X.
PUB. COUNTRY:        United States
DOCUMENT TYPE:       Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:            English
FILE SEGMENT:        Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH:         200503
ENTRY DATE:          Entered STN: 20050209
                     Last Updated on STN: 20050325
                     Entered Medline: 20050324
AB   Hypothalamic GnRH is a decapeptide that plays a pivotal role in mammalian
     reproduction by stimulating the synthesis and secretion of gonadotropins
     via binding to the GnRH receptor on the pituitary gonadotropins.  It is
     hypothesized that sex steroids may regulate GnRH I (a classical form of
     GnRH), GnRH II (a second form of GnRH), and GnRH I receptor (GnRHRI) at
     the transcriptional level in target tissues.  Thus, in the present study a
     role for progesterone (P4) in the regulation of GnRH I, GnRH II, and
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GnRHRI was investigated using a human neuronal **medulloblastoma** cell line (TE671) as an in vitro model. The cells were transfected with human GnRHRI promoter-luciferase constructs, and promoter activities were analyzed after P4 treatment by luciferase and beta-galactosidase assay. The mRNA levels of GnRH I and GnRH II were analyzed by RT-PCR. Treatment of TE671 cells with P4 resulted in a decrease in GnRHRI promoter activity compared with the control level in a dose- and time-dependent manner. Cotreatment of these cells with RU486, an antagonist of P4, reversed P4-induced inhibition of GnRHRI promoter activity, suggesting that the P4 effect is mediated by P4 receptor (PR). In the cells transfected with a full-length of PR A- or PR B-expressing vector, overexpression of PR A increased the sensitivity toward P4 in an inhibition of GnRHRI promoter, whereas PR B increased transcriptional activity of GnRHRI promoter in the presence of P4. However, PR B itself did not act as a transcriptional activator of GnRHRI promoter. Because TE671 cells have been recently demonstrated to express and synthesize two forms of GnRHs, we also investigated the regulation of GnRH mRNAs by P4. In the present study, P4 increased GnRH I mRNA levels in a time- and dose-dependent manner. This stimulatory effect of P4 in the regulation of GnRH I mRNAs was significantly attenuated by RU486, whereas no significant difference in the expression level of GnRH II was observed with P4 or RU496. Interestingly, although the expression level of PR B was low compared with that of PR A, P4 action on the GnRH I gene was mediated by PR B. In conclusion, these results indicate that P4 is a potent regulator of GnRHRI at the transcriptional level as well as GnRH I mRNA. This distinct effect of P4 on the GnRH system may be derived from different pathways through PR A or PR B.

L5 ANSWER 2 OF 6 MEDLINE on STN
ACCESSION NUMBER: 2002198301 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11931351
TITLE: The transcription of the hGnRH-I and hGnRH-II genes in human neuronal cells is differentially regulated by estrogen.
AUTHOR: Chen Alon; Zi Keren; Laskar-Levy Orly; Koch Yitzhak
CORPORATE SOURCE: Department of Neurobiology, Weizmann Institute of Science, Rehovot, Israel.
SOURCE: Journal of molecular neuroscience : MN, (2002 Feb-Apr) 18 (1-2) 67-76.
Journal code: 9002991. ISSN: 0895-8696.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200211
ENTRY DATE: Entered STN: 20020405
Last Updated on STN: 20021211
Entered Medline: 20021108

AB **Gonadotropin releasing hormone**-I (GnRH-I), a decapeptide serves as a key regulator of reproduction. Recently, several groups have identified in the mammalian brain a second form of GnRH, of unknown function, designated GnRH-II. The human neuronal **medulloblastoma** cells (TE-671) were recently demonstrated to express the two forms of GnRH (GnRH-I and GnRH-II). We used this cell line, as a model system, to investigate the regulation of human GnRH-I and GnRH-II genes by estrogen. Estrogen is one of the principal regulators of GnRH-I in hypothalamic neurons, acting as a classic homeostatic feedback molecule between the gonads and the brain. In this study, we investigated the regulation of the two GnRH forms by estrogen, in the human neuronal cell line TE-671. We demonstrate, for the first time, that the hGnRH-II and hGnRH-I genes are differentially regulated by estrogen. Using reverse transcriptase-polymerase chain reaction (RT-PCR) and Southern hybridization, we found that estrogen increases endogenous hGnRH-II mRNA levels and decreases endogenous hGnRH-I mRNA levels. Furthermore, we found these effects to be promoter-mediated. We cloned the hGnRH-I and

hGnRH-II promoter constructs upstream to a luciferase reporter plasmid, and cotransfected these constructs with an estrogen receptor alpha into the TE-671 neuronal cells. Luciferase activity of GnRH promoter constructs treated with estrogen demonstrates that the differential regulation of the GnRH genes by estrogen is mediated at the transcription level.

L5 ANSWER 3 OF 6 MEDLINE on STN
ACCESSION NUMBER: 2001179519 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11159856
TITLE: Two isoforms of **gonadotropin-releasing hormone** are coexpressed in neuronal cell lines.
AUTHOR: Chen A; Yahalom D; Laskar-Levy O; Rahimipour S; Ben-Aroya N; Koch Y
CORPORATE SOURCE: Department of Neurobiology, Weizmann Institute of Science, Rehovot 76100, Israel.
SOURCE: Endocrinology, (2001 Feb) 142 (2) 830-7.
Journal code: 0375040. ISSN: 0013-7227.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200103
ENTRY DATE: Entered STN: 20010404
Last Updated on STN: 20010404
Entered Medline: 20010329

AB GnRH-I serves as the neuropeptide that regulates mammalian reproduction. Recently, several groups have identified in the brain of rodents, monkeys, and humans a second isoform of GnRH (GnRH-II) whose structure is 70% identical to that of GnRH-I. In this study we demonstrate for the first time human and mouse neuronal cell lines that express both GnRH-I and GnRH-II. Following the screening of several human neuronal cell lines by RT-PCR and Southern hybridization, we demonstrated that two cell lines, TE-671 **medulloblastoma** and LAN-1 neuroblastoma cells, coexpress messenger RNA encoding the two isoforms of GnRH. Nucleotide sequencing indicated that the complementary DNA fragments are identical to those of the known human GnRH-I and GnRH-II sequences. Extracts obtained from the TE-671 and LAN-1 cell lines as well as from the immortalized mouse hypothalamic GT1-7 neuronal cell line were found to contain the two isoforms of GnRH, which exhibited identical chromatographic properties as synthetic GnRH-I and GnRH-II, in HPLC followed by specific RIAs. Furthermore, double immunofluorescence studies demonstrated the two GnRH isoforms in LAN-1, TE-671, and GT1-7 cells. The identification of neuronal cell lines expressing both GnRH-I and GnRH-II provides tools for studying the differential regulation of gene expression and secretion and for studying the interaction between the two isoforms. Such studies may contribute to elucidation of the physiological functions of GnRH-II, which are still unknown.

L5 ANSWER 4 OF 6 MEDLINE on STN
ACCESSION NUMBER: 2001050966 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11033292
TITLE: A case of atypical absence seizures induced by leuprolide acetate.
AUTHOR: Akaboshi S; Takeshita K
CORPORATE SOURCE: Division of Child Neurology, Institute of Neurological Sciences, Faculty of Medicine, Tottori University, Yonago, Japan.
SOURCE: Pediatric neurology, (2000 Sep) 23 (3) 266-8.
Journal code: 8508183. ISSN: 0887-8994.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CASE REPORTS)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals

ENTRY MONTH: 200012
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001211

AB We report a case of a 13-year-old female with atypical absence seizures induced by prolonged administration of long-acting leuprolide acetate (LA). This patient had brain involvement resulting from chemotherapy and radiotherapy for a **medulloblastoma**. At 13 years of age, administration of long-acting LA was started. After the third dose of long-acting LA, atypical absence seizures appeared. After discontinuing long-acting LA, the seizures stopped without administration of any antiepileptic drugs. However, 2 years, 6 months later, the same seizures again appeared. On the basis of the findings of endocrinologic investigations and the reported data of pharmacokinetics of LA, we speculate that her seizures were induced by LA and that the seizures were associated with the presence of brain damage in the patient. Care should therefore be taken when using long-acting LA or other **gonadotropin -releasing hormone** analogues for pediatric patients with diffuse brain damage.

L5 ANSWER 5 OF 6 MEDLINE on STN
ACCESSION NUMBER: 2000079233 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10611579
TITLE: Adult height after growth hormone (GH) treatment for GH deficiency due to cranial irradiation.
AUTHOR: Adan L; Sainte-Rose C; Souberbielle J C; Zucker J M; Kalifa C; Brauner R
CORPORATE SOURCE: Pediatric Endocrinology Department, Universite Rene Descartes and Hopital Necker-Enfants Malades, Assistance Publique-Hopitaux de Paris, Paris, France.
SOURCE: Medical and pediatric oncology, (2000 Jan) 34 (1) 14-9. Journal code: 7506654. ISSN: 0098-1532.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200002
ENTRY DATE: Entered STN: 20000209
Last Updated on STN: 20000209
Entered Medline: 20000203

AB BACKGROUND: The indications and factors affecting the growth in response to treatment with growth hormone (GH) of patients with cranial irradiation-induced GH deficiency remain unclear. PROCEDURE: The adult heights of 56 patients treated with GH (0.4-0.6 U/kg/week) as daily sc injections were analysed. They had been given 18 or 24 Grays (Gy) cranial irradiation for leukemia (group 1, 26 cases), 50 +/- 1 Gy for various tumors (group 2, 13 cases), 46 +/- 1 Gy for retinoblastoma (group 3, 8 cases), or 34 +/- 2 Gy with spinal irradiation for **medulloblastoma** (group 4, 9 cases). Twenty- five of these 56 patients had early puberty and were also treated with **gonadotropin-releasing hormone** (GnRH) analog. RESULTS: The standing (-1.0 +/- 0.2 in group 1, -0.7 +/- 0.3 in group 2, -1.1 +/- 0.3 in group 3, and -2.0 +/- 0.4 SD in group 4) and sitting (-1.8 +/- 0.2 in group 1, -0.4 +/- 0.4 in group 2, -1.2 +/- 0.4 in group 3, and -3. 4 +/-0.4 SD in group 4) adult heights were shor ter ($P < 0.05$ for standing and $P < 0.001$ for sitting heights) for group 4 than for each of the other groups. Of the 47 patients given cranial (and not craniospinal) irradiation, sitting adult height was shorter ($P = 0. 02$) and the difference between standing adult and target heights greater ($P = 0.03$) in those patients in whom puberty occurred at a normal age than in those treated with GnRH analog. Conclusion. The incomplete catch-up of growth seems to be mainly due to the reduction in sitting height of patients given spinal irradiation and in whom puberty occurred at a normal age. This suggests that GnRH analog treatment should be more widely used to treat children with early and/or

rapidly progressing puberty after cranial irradiation.
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L5 ANSWER 6 OF 6 MEDLINE on STN
ACCESSION NUMBER: 1999258039 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10326189
TITLE: Effects of puberty on bone age maturation in a girl after **medulloblastoma** therapy.
AUTHOR: Marx M; Schoof E; Grabenbauer G G; Beck J D; Doerr H G
CORPORATE SOURCE: Division of Paediatric Endocrinology, University of Erlangen-Nuremberg, Germany.
SOURCE: Journal of pediatric and adolescent gynecology, (1999 May) 12 (2) 62-6.
Journal code: 9610774. ISSN: 1083-3188.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CASE REPORTS)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199906
ENTRY DATE: Entered STN: 19990714
Last Updated on STN: 19990714
Entered Medline: 19990629

AB BACKGROUND: Craniospinal radiotherapy for malignant brain tumors can result in a variety of neuroendocrine disturbances, among which are the development of growth hormone deficiency and early puberty, which can markedly reduce adult height. METHODS: The authors report the case of a girl who received craniospinal radiotherapy for a **medulloblastoma** at the age of 3.4 years. At 9.1 years, growth hormone therapy was started, and spontaneous onset of puberty (Tanner stage B2) occurred at age 10.3 years. Interval until menarche was short, at only 0.9 years. RESULTS: Although chronologic age at appearance of Tanner stages was within the normal range, the patient showed a rapid acceleration in skeletal maturation, resulting in adult short stature. CONCLUSION: Bone age seems to be a more precise parameter for biologic maturation in some patients after craniospinal irradiation than is clinical assessment of pubertal stages. Thus, if progression of bone age and decreasing final height predictions are noted, puberty should be stopped with **gonadotropin-releasing hormone** analogs, even if pubertal development seems to be adequate for chronologic age, because this increases the remaining time for growth hormone treatment.

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NEWS 12	NOV 30	REGISTRY/ZREGISTRY on STN(R) enhanced with experimental spectral property data
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=> file medline
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FULL ESTIMATED COST	0.21	0.21

FILE 'MEDLINE' ENTERED AT 09:40:08 ON 10 JAN 2006

FILE LAST UPDATED: 7 JAN 2006 (20060107/UP). FILE COVERS 1950 TO DATE.

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NEWS HOURS	STN Operating Hours Plus Help Desk Availability
NEWS INTER	General Internet Information
NEWS LOGIN	Welcome Banner and News Items
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NEWS WWW	CAS World Wide Web Site (general information)

Enter NEWS followed by the item number or name to see news on that
specific topic.

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*IFICDB - The IFI Comprehensive Database from 1950-present
*IFIPAT - The IFI Patent Database from 1950-present
*IFIUDB - The IFI Uniterm Database from 1950-present

* The files listed above are temporarily unavailable.

FILE 'HOME' ENTERED AT 09:42:34 ON 10 JAN 2006

=> file medline
COST IN U.S. DOLLARS

	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'MEDLINE' ENTERED AT 09:42:59 ON 10 JAN 2006

FILE LAST UPDATED: 7 JAN 2006 (20060107/UP). FILE COVERS 1950 TO DATE.

On December 11, 2005, the 2006 MeSH terms were loaded.

The MEDLINE reload for 2006 will soon be available. For details on the 2005 reload, enter HELP RLOAD at an arrow promt (=>). See also:

http://www.nlm.nih.gov/mesh/
http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html
http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_med_data_changes.html
http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_2006_MeSH.html

OLDMEDLINE is covered back to 1950.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2006 vocabulary.

This file contains CAS Registry Numbers for easy and accurate

```
=> s tu GnRH or gonadotropin(w)releasing(w)hormone
       1145754 TU
           204 TUS
       1145918 TU
                 (TU OR TUS)
         11745 GNRH
           107 GNRHS
         11747 GNRH
                 (GNRH OR GNRHS)
             0 TU GNRH
                 (TU(W)GNRH)
         41701 GONADOTROPIN
         22719 GONADOTROPINS
         55507 GONADOTROPIN
                 (GONADOTROPIN OR GONADOTROPINS)
         57465 RELEASING
        268964 HORMONE
        177879 HORMONES
        389158 HORMONE
                 (HORMONE OR HORMONES)
```

```
          7655 GONADOTROPIN(W)RELEASING(W)HORMONE
L1        7655 TU GNRH OR GONADOTROPIN(W)RELEASING(W)HORMONE

=> s l1 and pinealoma
          1238 PINEALOMA
            51 PINEALOMAS
          1251 PINEALOMA
                 (PINEALOMA OR PINEALOMAS)
L2           0 L1 AND PINEALOMA

=> s l1 and neuroblastoma
         22738 NEUROBLASTOMA
          2006 NEUROBLASTOMAS
         23167 NEUROBLASTOMA
                 (NEUROBLASTOMA OR NEUROBLASTOMAS)
L3           3 L1 AND NEUROBLASTOMA

=> dis ibib abs l2 1-3
L2 HAS NO ANSWERS
L1         7655 SEA FILE=MEDLINE ABB=ON  PLU=ON  TU GNRH OR GONADOTROPIN(W)RELE
                ASING(W)HORMONE
L2            0 SEA FILE=MEDLINE ABB=ON  PLU=ON  L1 AND PINEALOMA


=> dis ibib abs l3 1-3

L3   ANSWER 1 OF 3     MEDLINE on STN
ACCESSION NUMBER:   2003030963     MEDLINE
DOCUMENT NUMBER:    PubMed ID: 12538601
TITLE:              A transcriptionally active human type II
                    gonadotropin-releasing hormone
                    receptor gene homolog overlaps two genes in the antisense
                    orientation on chromosome 1q.12.
AUTHOR:             Morgan Kevin; Conklin Darrell; Pawson Adam J; Sellar Robin;
                    Ott Thomas R; Millar Robert P
CORPORATE SOURCE:   Medical Research Council Human Reproductive Sciences Unit,
                    University of Edinburgh Academic Centre, Edinburgh EH16
                    4SB, United Kingdom.. k.morgan@hrsu.mrc.ac.uk
SOURCE:             Endocrinology, (2003 Feb) 144 (2) 423-36.
                    Journal code: 0375040. ISSN: 0013-7227.
PUB. COUNTRY:       United States
DOCUMENT TYPE:      Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:           English
FILE SEGMENT:       Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH:        200302
ENTRY DATE:         Entered STN: 20030123
                    Last Updated on STN: 20030214
                    Entered Medline: 20030213
AB   GnRH-II peptide hormone exhibits complete sequence conservation across
     vertebrate species, including man.  Type-II GnRH receptor genes have been
     characterized recently in nonhuman primates, but the human receptor gene
     homolog contains a frameshift, a premature stop codon (UGA), and a 3'
     overlap of the RBM8A gene on chromosome 1q.12.  A retrotransposed
     pseudogene, RBM8B, retains partial receptor sequence.  In this study,
     bioinformatics show that the human receptor gene promoter overlaps the
     peroxisomal protein 11-beta gene promoter and the premature UGA is
     positionally conserved in chimpanzee.  A CGA [arginine (Arg)] occurs in
     porcine DNA, but UGA is shifted one codon to the 5' direction in bovine
     DNA, suggesting independent evolution of premature stop codons.  In
     contrast to marmoset tissue RNA, exon- and strand-specific probes are
     required to distinguish differently spliced human receptor gene
     transcripts in cell lines (HP75, IMR-32).  RBM8B is not transcribed.
     Sequencing of cDNAs for spliced receptor mRNAs showed no evidence for
     alteration of the premature UGA by RNA editing, but alternative splicing
```

circumvents the frameshift to encode a two-membrane-domain protein before this UGA. A stem-loop motif resembling a selenocysteine insertion sequence and a potential alternative translation initiation site might enable expression of further proteins involved in interactions within the GnRH system.

L3 ANSWER 2 OF 3 MEDLINE on STN
ACCESSION NUMBER: 2001179519 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11159856
TITLE: Two isoforms of **gonadotropin-releasing hormone** are coexpressed in neuronal cell lines.
AUTHOR: Chen A; Yahalom D; Laskar-Levy O; Rahimipour S; Ben-Aroya N; Koch Y
CORPORATE SOURCE: Department of Neurobiology, Weizmann Institute of Science, Rehovot 76100, Israel.
SOURCE: Endocrinology, (2001 Feb) 142 (2) 830-7.
Journal code: 0375040. ISSN: 0013-7227.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200103
ENTRY DATE: Entered STN: 20010404
Last Updated on STN: 20010404
Entered Medline: 20010329

AB GnRH-I serves as the neuropeptide that regulates mammalian reproduction. Recently, several groups have identified in the brain of rodents, monkeys, and humans a second isoform of GnRH (GnRH-II) whose structure is 70% identical to that of GnRH-I. In this study we demonstrate for the first time human and mouse neuronal cell lines that express both GnRH-I and GnRH-II. Following the screening of several human neuronal cell lines by RT-PCR and Southern hybridization, we demonstrated that two cell lines, TE-671 medulloblastoma and LAN-1 **neuroblastoma** cells, coexpress messenger RNA encoding the two isoforms of GnRH. Nucleotide sequencing indicated that the complementary DNA fragments are identical to those of the known human GnRH-I and GnRH-II sequences. Extracts obtained from the TE-671 and LAN-1 cell lines as well as from the immortalized mouse hypothalamic GT1-7 neuronal cell line were found to contain the two isoforms of GnRH, which exhibited identical chromatographic properties as synthetic GnRH-I and GnRH-II, in HPLC followed by specific RIAs. Furthermore, double immunofluorescence studies demonstrated the two GnRH isoforms in LAN-1, TE-671, and GT1-7 cells. The identification of neuronal cell lines expressing both GnRH-I and GnRH-II provides tools for studying the differential regulation of gene expression and secretion and for studying the interaction between the two isoforms. Such studies may contribute to elucidation of the physiological functions of GnRH-II, which are still unknown.

L3 ANSWER 3 OF 3 MEDLINE on STN
ACCESSION NUMBER: 81142294 MEDLINE
DOCUMENT NUMBER: PubMed ID: 6259147
TITLE: Interaction of fluorescent **gonadotropin-releasing hormone** with receptors in cultured pituitary cells.
AUTHOR: Naor Z; Atlas D; Clayton R N; Forman D S; Amsterdam A; Catt K J
SOURCE: Journal of biological chemistry, (1981 Mar 25) 256 (6) 3049-52.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198105

ENTRY DATE: Entered STN: 19900316
Last Updated on STN: 19970203
Entered Medline: 19810526

AB A fluorescent derivative of the **gonadotropin-releasing hormone** (GnRH) agonist analog, [D-Lys6]GnRH, was synthesized for receptor studies and shown to be biologically active. The rhodamine-derivatized peptide (Rh-GnRH) retained 40% of the receptor binding activity of [D-Lys6]GnRH, and 50% of the luteinizing hormone-releasing activity assayed in cultured pituitary cells. The fluorescent analog was employed to visualize the distribution of GnRH receptors in cultured pituitary cells, using the technique of video-intensified fluorescence microscopy. The binding of Rh-GnRH was confined to the large gonadotrophs which comprised 15% of the cell population. The specificity of the binding was shown by the absence of significant fluorescence in the presence of a 100-fold excess of [D-Lys6]GnRH, or when Rh-GnRH was incubated with choriocarcinoma, **neuroblastoma**, or 3T3 cell lines devoid of GnRH receptors. The interaction of Rh-GnRH with living pituitary cells was characterized by an initial diffuse distribution, followed by the formation of polar aggregates that later appeared to be internalized. These observations emphasize the value of fluorescent derivatives of GnRH for elucidating the course of the interaction with specific receptors on pituitary gonadotrophs. The initial results indicate that GnRH-receptor complexes undergo aggregation during stimulation of luteinizing hormone release, and are later internalized for subsequent degradation and/ or intracellular actions.

=> s l1 and craniopharyngeoma
24 CRANIOPHARYNGEOMA
20 CRANIOPHARYNGEOMAS
43 CRANIOPHARYNGEOMA
(CRANIOPHARYNGEOMA OR CRANIOPHARYNGEOMAS)
L4 1 L1 AND CRANIOPHARYNGEOMA

=> dis ibib abs l4

L4 ANSWER 1 OF 1 MEDLINE on STN
ACCESSION NUMBER: 91141849 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1996204
TITLE: [Pulsatile **gonadotropin-releasing hormone** substitution following excision of a craniopharyngioma with suprasellar invasion]. Pulzatorikus gonadotropin releasing hormon substitutio suprasellaris novekedesu **craniopharyngeoma** eltavolitasat kovetoen.
AUTHOR: Koloszar S; Bartfai G; Sas M
CORPORATE SOURCE: Szuleszeti es Nogyogyaszati Klinika, Szent-Gyorgi Albert Orvostudomanyi Egyetem, Szeged.
SOURCE: Orvosi hetilap, (1991 Jan 20) 132 (3) 139-41.
Journal code: 0376412. ISSN: 0030-6002.
PUB. COUNTRY: Hungary
DOCUMENT TYPE: (CASE REPORTS)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Hungarian
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199103
ENTRY DATE: Entered STN: 19910412
Last Updated on STN: 19910412
Entered Medline: 19910326

AB **Craniopharyngeoma** growing suprasellary attacks the medio-basal region of hypothalamus, that leads to the stopping of the production of **gonadotropin releasing hormone**. In connection with the case of a 15-year-old girl who had partial extirpation of

craniopharyngeoma the authors write about the favourable endocrine effect of pulsatile **gonadotropin releasing hormone** treatment. Through giving **gonadotropin releasing hormone** every 90 minutes in 20 micrograms doses menstruation cycle and ovulation was performed. Beside surgical treatment hormonal substitution plays an important role in the treatment of additional endocrine symptoms.

```
=> s l1 and meningeoma
            93 MENINGEOMA
            56 MENINGEOMAS
           141 MENINGEOMA
                 (MENINGEOMA OR MENINGEOMAS)
L5           0 L1 AND MENINGEOMA

=> s l1 and chordoma
          2211 CHORDOMA
           636 CHORDOMAS
          2301 CHORDOMA
                 (CHORDOMA OR CHORDOMAS)
L6           0 L1 AND CHORDOMA

=> s l1 and Ewing sarcoma
          5448 EWING
            54 EWINGS
          5483 EWING
                 (EWING OR EWINGS)
         70545 SARCOMA
         11081 SARCOMAS
           119 SARCOMATA
         73808 SARCOMA
                 (SARCOMA OR SARCOMAS OR SARCOMATA)
           769 EWING SARCOMA
                 (EWING(W)SARCOMA)
L7           0 L1 AND EWING SARCOMA

=> s l1 and malignant(w)melanoma
        191227 MALIGNANT
             9 MALIGNANTS
        191228 MALIGNANT
                 (MALIGNANT OR MALIGNANTS)
         60376 MELANOMA
          9262 MELANOMAS
            80 MELANOMATA
             1 MELANOMATAS
         61358 MELANOMA
                 (MELANOMA OR MELANOMAS OR MELANOMATA OR MELANOMATAS)
         16936 MALIGNANT(W)MELANOMA
L8           0 L1 AND MALIGNANT(W)MELANOMA

=> s l1 and Kaposi(w)sarcoma
         10558 KAPOSI
            40 KAPOSIS
         10568 KAPOSI
                 (KAPOSI OR KAPOSIS)
         70545 SARCOMA
         11081 SARCOMAS
           119 SARCOMATA
         73808 SARCOMA
                 (SARCOMA OR SARCOMAS OR SARCOMATA)
          1041 KAPOSI(W)SARCOMA
L9           0 L1 AND KAPOSI(W)SARCOMA
```

```
=> s l1 and (brain(w)tumor or meningeal(w)tumor)
         661971 BRAIN
          26207 BRAINS
         667265 BRAIN
                  (BRAIN OR BRAINS)
         625474 TUMOR
         271064 TUMORS
         747795 TUMOR
                  (TUMOR OR TUMORS)
          14457 BRAIN(W)TUMOR
          18967 MENINGEAL
              1 MENINGEALS
          18967 MENINGEAL
                  (MENINGEAL OR MENINGEALS)
         625474 TUMOR
         271064 TUMORS
         747795 TUMOR
                  (TUMOR OR TUMORS)
            131 MENINGEAL(W)TUMOR
L10           7 L1 AND (BRAIN(W)TUMOR OR MENINGEAL(W)TUMOR)

=> dup rem
ENTER L# LIST OR (END):l10
PROCESSING COMPLETED FOR L10
L11           7 DUP REM L10 (0 DUPLICATES REMOVED)

=> dis ibib abs l11 1-7

L11  ANSWER 1 OF 7     MEDLINE on STN
ACCESSION NUMBER:   2002464597     MEDLINE
DOCUMENT NUMBER:    PubMed ID: 12182973
TITLE:              Preirradiation endocrinopathies in pediatric brain
                    tumor patients determined by dynamic tests of
                    endocrine function.
AUTHOR:             Merchant Thomas E; Williams Tani; Smith Julie M; Rose Susan
                    R; Danish Robert K; Burghen George A; Kun Larry E; Lustig
                    Robert H
CORPORATE SOURCE:   Department of Radiation Oncology, St. Jude Children's
                    Research Hospital, 332 North Lauderdale Street, Memphis, TN
                    38105, USA.. thomas.merchant@stjude.org
CONTRACT NUMBER:    P30 CA 21765 (NCI)
SOURCE:             International journal of radiation oncology, biology,
                    physics, (2002 Sep 1) 54 (1) 45-50.
                    Journal code: 7603616. ISSN: 0360-3016.
PUB. COUNTRY:       United States
DOCUMENT TYPE:      Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:           English
FILE SEGMENT:       Priority Journals
ENTRY MONTH:        200209
ENTRY DATE:         Entered STN: 20020913
                    Last Updated on STN: 20020927
                    Entered Medline: 20020926
AB   PURPOSE: To prospectively evaluate pediatric patients with localized
     primary brain tumors for evidence of endocrinopathy
     before radiotherapy (RT). METHODS AND MATERIALS: Seventy-five pediatric
     patients were evaluated with the arginine tolerance test and L-dopa test
     for growth hormone secretory capacity and activity; thyroid-stimulating
     hormone surge and thyrotropin-releasing hormone stimulation test for the
     hypothalamic-thyroid axis; the 1-microg adrenocorticotropin hormone (ACTH)
     and metyrapone test for ACTH reserve; and, depending on age, a
     gonadotropin-releasing hormone stimulation
     test to determine gonadotropin response. The study included 38 male and
     37 female patients, age 1-21 years with ependymoma (n = 35), World Health
     Organization (WHO) Grade I-II astrocytoma (n = 18), WHO Grade III-IV
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astrocytoma (n = 10), craniopharyngioma (n = 7), optic pathway tumor (n = 4), and germinoma (n = 1). Seven patients receiving dexamethasone at the time of the evaluation were excluded from the final analysis. RESULTS: Of 68 assessable patient, 45 (66%) had evidence of endocrinopathy before RT, including 15 of 32 patients (47%) with posterior fossa tumors. Of the 45 patients, 38% had growth hormone deficiency, 43% had thyroid-stimulating hormone secretion abnormality, 22% had an abnormality in ACTH reserve, and 13% had an abnormality in age-dependent gonadotropin secretion. CONCLUSION: The incidence of pre-RT endocrinopathy in pediatric **brain tumor** patients is high, including patients with tumors not adjacent to the hypothalamic-pituitary unit. These data suggest an overestimation in the incidence of radiation-induced endocrinopathy. Baseline endocrine function should be determined for **brain tumor** patients before therapy. The potential for radiation-induced endocrinopathy alone cannot be used as an argument for alternatives to RT for most patients. Pre-RT endocrinopathy may be an early indicator of central nervous system damage that will influence the functional outcome unrelated to RT.

L11 ANSWER 2 OF 7 MEDLINE on STN
ACCESSION NUMBER: 2001518088 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11528557
TITLE: [Cranial irradiation induces premature activation of the **gonadotropin-releasing-hormone**].
Schadelbestrahlung verursacht vorzeitige Aktivierung des Gonadotropin-Releasinghormon (GnRH)-Pulsgenerators bei Ratten - Ein neuesTiermodell fur strahleninduzierte Storungen der Pubertat.
AUTHOR: Roth C; Lakomek M; Schmidberger H; Jarry H
CORPORATE SOURCE: Kinderklinik, Germany.
SOURCE: Klinische Padiatrie, (2001 Jul-Aug) 213 (4) 239-43.
Journal code: 0326144. ISSN: 0300-8630.
PUB. COUNTRY: Germany: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200112
ENTRY DATE: Entered STN: 20010924
Last Updated on STN: 20020122
Entered Medline: 20011204

AB BACKGROUND: CNS-irradiation in prepubertal children with leukemia or **brain tumors** can lead to precocious or in high doses to delayed puberty. The underlying mechanisms of these disorders are unknown. METHODS: A new animal model of experimentally induced pubertal disorders by cranial irradiation has been developed. In infantile or juvenile (12 - 23 days old) female rats precocious or delayed puberty have been induced by selective cranial Co60-irradiation (4 - 18 Gy). At age of 32 - 38 days or 3 months relevant hormone parameters have been studied basal and after stimulated conditions. RESULTS: Low radiation doses (5 or 6 Gy) led to accelerated onset of puberty as well as elevated LH- and estradiol levels. High radiation doses (9 - 18 Gy) caused retardation of sexual development, lower gonadotropin levels and growth retardation associated with growth hormone deficiency. After cranial irradiation with 5 Gy the release rates of the inhibitory neurotransmitter gamma-aminobutyric-acid (GABA) from hypothalamic explants were significantly lower ($p < 0,05$). The **gonadotropin-releasing-hormone** (GnRH) expression in the hypothalamic preoptic area of irradiated animals (5 Gy) was significantly higher than in controls ($p < 0,05$). CONCLUSION: The GnRH-pulse generator is very radiosensitive as low dose irradiation causes precocious puberty, whereas high dose irradiation is associated with delayed sexual maturation. Radiation induced precocious puberty might be caused by damage to inhibitory GABAergic neurons leading to desinhibition and premature

activation of GnRH neurons. Our animal model of cranial irradiation seems to be suitable to study neurotransmitter disorders, molecular mechanisms and potential preventive intervention of radiation induced pubertal changes.

L11 ANSWER 3 OF 7 MEDLINE on STN
ACCESSION NUMBER: 1999258039 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10326189
TITLE: Effects of puberty on bone age maturation in a girl after medulloblastoma therapy.
AUTHOR: Marx M; Schoof E; Grabenbauer G G; Beck J D; Doerr H G
CORPORATE SOURCE: Division of Paediatric Endocrinology, University of Erlangen-Nuremberg, Germany.
SOURCE: Journal of pediatric and adolescent gynecology, (1999 May) 12 (2) 62-6.
Journal code: 9610774. ISSN: 1083-3188.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CASE REPORTS)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199906
ENTRY DATE: Entered STN: 19990714
Last Updated on STN: 19990714
Entered Medline: 19990629

AB BACKGROUND: Craniospinal radiotherapy for malignant **brain tumors** can result in a variety of neuroendocrine disturbances, among which are the development of growth hormone deficiency and early puberty, which can markedly reduce adult height. METHODS: The authors report the case of a girl who received craniospinal radiotherapy for a medulloblastoma at the age of 3.4 years. At 9.1 years, growth hormone therapy was started, and spontaneous onset of puberty (Tanner stage B2) occurred at age 10.3 years. Interval until menarche was short, at only 0.9 years. RESULTS: Although chronologic age at appearance of Tanner stages was within the normal range, the patient showed a rapid acceleration in skeletal maturation, resulting in adult short stature. CONCLUSION: Bone age seems to be a more precise parameter for biologic maturation in some patients after craniospinal irradiation than is clinical assessment of pubertal stages. Thus, if progression of bone age and decreasing final height predictions are noted, puberty should be stopped with **gonadotropin-releasing hormone** analogs, even if pubertal development seems to be adequate for chronologic age, because this increases the remaining time for growth hormone treatment.

L11 ANSWER 4 OF 7 MEDLINE on STN
ACCESSION NUMBER: 1998419995 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9749566
TITLE: Changes in peripheral blood levels and pulse frequencies of GnRH in patients with hypopituitarism.
AUTHOR: Hayashi M; Takanashi N; Yaoi Y
CORPORATE SOURCE: Department of Obstetrics and Gynecology, Koshigaya Hospital, Dokkyo University School of Medicine, Japan.. mhayashi@dokkyomed.ac.jp
SOURCE: American journal of the medical sciences, (1998 Sep) 316 (3) 213-9.
Journal code: 0370506. ISSN: 0002-9629.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL)
(CONTROLLED CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199810

ENTRY DATE: Entered STN: 19981020
Last Updated on STN: 19981020
Entered Medline: 19981007

AB Pituitary dysfunction occasionally results from **brain tumors** or the surgical resection of **brain tumors** . The authors examined two patients with hypogonadotropic secondary amenorrhea, who had undergone surgical removal of **brain tumors**. Changes in immunoreactive **gonadotropin-releasing hormone** (GnRH) secretion are of interest in patients with a gonadotropin and gonadal steroid deficit, because both steroid and pituitary feedback systems are altered by tumors or tumor resection. The authors thus measured GnRH, luteinizing hormone, and follicle-stimulating hormone levels every 15 minutes for 4 hours by radioimmunoassay and investigated qualitative and quantitative changes in the pulsatile patterns of these hormones in two hypogonadotropic hypogonadism patients. They also performed similar multiple measurements of GnRH in two normal cycle women in follicular phase and two postmenopausal women. The concentration of plasma GnRH in two hypopituitarism patients was compared with that in two normal cycle women and two postmenopausal women. The study showed that the peripheral blood level of GnRH was significantly lower in two hypopituitarism patients than in both normal cycle and postmenopausal women, and that the pulsatile frequency was not different among these three groups. These findings suggest that alteration of feedback systems results in a decrease in the blood level of GnRH, and that pulses of GnRH maintain normal fluctuation despite the alteration of the hormonal circumstances in two hypogonadotropic hypogonadism patients.

L11 ANSWER 5 OF 7 MEDLINE on STN
ACCESSION NUMBER: 97284740 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9139717
TITLE: An alternative **gonadotropin-releasing hormone** (GnRH) RNA splicing product found in cultured GnRH neurons and mouse hypothalamus.
AUTHOR: Zhen S; Dunn I C; Wray S; Liu Y; Chappell P E; Levine J E; Radovick S
CORPORATE SOURCE: Department of Medicine, Division of Endocrinology, Children's Hospital, Harvard Medical School, Boston, Massachusetts 02115, USA.
CONTRACT NUMBER: HD30040 (NICHD)
SOURCE: Journal of biological chemistry, (1997 May 9) 272 (19) 12620-5.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199706
ENTRY DATE: Entered STN: 19970630
Last Updated on STN: 20021015
Entered Medline: 19970616

AB **Gonadotropin-releasing hormone** (GnRH) is encoded by the proGnRH gene which contains four exons and three introns. In this study, two immortalized GnRH-expressing cell lines (Gn11 and NLT) were characterized. The NLT and Gn11 cells, derived from a same **brain tumor** in a transgenic mouse, display neuronal morphology and neuron-specific markers. However, NLT cells secrete much higher levels of GnRH than Gn11 cells. To delineate the mechanism underlying this difference, reverse transcriptase-polymerase chain reaction and RNase protection assays were performed to examine proGnRH gene expression. While the mature proGnRH mRNA was predominately expressed in NLT cells, Gn11 cells express an abundant short transcript. Sequence analysis revealed that this short transcript contains exons 1, 3, and 4, but not exon 2, which encodes the GnRH decapeptide. RNase

protection assays demonstrated that NLT cells express much higher levels of mature proGnRH mRNA than Gn11 cells. The lower level of GnRH secreting capacity in Gn11 cells is due, in part, to decreased expression of mature proGnRH mRNA. When proGnRH gene expression in the mouse brain was examined, the same short splicing variant was observed in the olfactory area and preoptic area-anterior hypothalamus. But the prevalent transcript in these regions was the mature proGnRH mRNA. In contrast, only the mature proGnRH mRNA was found in the caudal hypothalamus. These results suggest that alternative splicing may be one of the mechanisms regulating proGnRH gene expression in the animal brain.

L11 ANSWER 6 OF 7 MEDLINE on STN
ACCESSION NUMBER: 97395596 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9251734
TITLE: Increased LH and FSH secretion after cranial irradiation in boys.
AUTHOR: Lannering B; Jansson C; Rosberg S; Albertsson-Wikland K
CORPORATE SOURCE: Department of Paediatrics, University of Goteborg, Sweden.
SOURCE: Medical and pediatric oncology, (1997 Oct) 29 (4) 280-7. Journal code: 7506654. ISSN: 0098-1532.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199709
ENTRY DATE: Entered STN: 19970916
Last Updated on STN: 19970916
Entered Medline: 19970903

AB The effect of high-dose cranial- and craniospinal irradiation and chemotherapy on the gonadotropin-sex steroid axis was studied during different stages of puberty by measuring pulsatile secretion of luteinizing hormone (LH), follicle-stimulating hormone (FSH) and testosterone. The patients were thirteen boys who had been treated for malignant **brain tumor** residing well away from the hypothalamo-pituitary region. The median time to follow-up was 9 (1-16) years. The onset of puberty was early in the patients, median 10.5 years, compared to the average age for Swedish boys, which is at median 12.4 years. There was, before puberty, no significant difference in LH and FSH secretion between patients and a control group of normal boys. In early, mid- and late stages of puberty, however, LH and FSH secretion was increased in the patients overall, whereas testosterone secretion was maintained within the normal range in spite of signs of gonadotoxocity with small testicular volumes. These results indicate that the vulnerable parts of the **gonadotropin releasing hormone** (GnRH)-gonadotropin (LH, FSH)-gonadal axis are the regulatory system that determines the timing of pubertal induction and the gonads. The GnRH-LH, FSH-releasing neurons appear relatively resistant to cranial irradiation as they are able to respond with supranormal LH and FSH levels for long periods of time after treatment.

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AUTHOR: Kitanaka C; Matsutani M; Sora S; Kitanaka S; Tanae A; Hibi I
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AB Although precocious puberty is common in boys with human chorionic gonadotropin (hCG)-secreting **brain tumors**, it is extremely rare in girls. The authors describe a 6-year-old girl with an hCG-secreting suprasellar immature teratoma who presented with diabetes insipidus, increased intracranial pressure, and precocious puberty. On admission, breast budding was observed. The serum hCG level was 1230 mIU/ml. Both luteinizing hormone (LH) and follicle-stimulating hormone (FSH) remained below detectable levels, even after **gonadotropin-releasing hormone** stimulation. Serum estrogen and androgen were moderately elevated. After chemotherapy, breast budding disappeared with normalization of serum hCG. It has been believed that hCG does not produce precocious puberty in girls in the absence of FSH, and this has been used as an explanation for the rarity of precocious puberty in girls with hCG-secreting **brain tumors**.
However, it has also been reported that hCG has not only LH activity but also intrinsic, although weak, FSH-like activity. In the present case, this FSH-like activity was considered to have played a role in the development of precocious puberty. It is speculated that a very high level of serum hCG can produce precocious puberty in girls. The rarity of intracranial germ-cell tumors with a high potential of hCG secretion may be one of the reasons why hCG-induced precocious puberty is uncommon in girls.